SOME PHASES OF GALLSTONE DISEASE, WITH SPECIAL REFERENCE TO DIAGNOSIS AND TREATMENT.

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THE dominant factor in the formation of gallstones apparently is bacterial invasion of the gall-bladder plus a tendency to stagnation of the bile in that viscus. It was formerly thought, since normal bile is usually sterile, bacteria could not thrive in that fluid; but it is now established that bile is a fertile culture medium for many varieties of pathogenic bacteria, notably the Eberth bacillus and the colon bacillus and its varieties, which may assume pathogenic properties.

The bacteria most commonly found in connection with cholecystitis and gallstone formation are the colon and typhoid bacillus, and, less commonly, the gall-bladder is invaded by streptococci, staphylococci, and the pneumococcus.[1]

The invasion of the gall-bladder by bacteria occurs either by the blood (portal vein, thence eliminated by the liver in the bile), the likely mode of the almost invariable infection of the gall-bladder in typhoid fever, or through the duodenum by means of the common bile duct and the cystic duct, the probable source of colon bacillus infection.

With the latter mode of entry the infection may and often is a mixed one, since the healthy duodenum is said to normally harbor not only the colon organism, but also staphylococci and streptococci. It seems established, as just remarked, that in nearly all, if not in all, cases of typhoid fever infection of the bile by the specific microorganism occurs. It is interesting, such being the case, that in only a limited number of these does cholecystitis immediately or subsequently develop, or are pathological changes in the walls of the gall-bladder found in cases of lethal typhoid.

Bacteria do not normally readily migrate into the bile passages from the intestines, and the pathological condition which favors their entrance is somewhat similar to that which favors, by their aid, gallstone formation—stagnation in the outflow of bile through the ducts. Obtaining an entrance into the gall-bladder, these organisms probably lead to the formation of gallstones in the manner originally suggested by Naunyn, by inducing a catarrh of the gall-bladder through infection of its wall; or even without such an infection the formation of calculi may be promptly favored by virtue of the acid production of such organisms as the Eberth and the colon bacillus. This acid production leads to precipitation of bile acids and of cholesterin. Stagnation of bile occurs in the gall-bladder, whence result concentration, and subsequently, through cholesterin, bile acids, and bilirubin-calcium precipitation, stone formation is readily induced.[1]

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[1] For details of this part of the subject the reader is referred to the article by Pratt, Typhoid Cholecystitis, with Observations upon Gallstone Formations, THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, November, 1891; and that of Richardson on the Bacteriology of the Gall-bladder, in Warren's Surgical Pathology and Therapeutics.

Micro-organisms have been found in many instances of fairly recent stone formations in the very centre of the stone, as if they had been an original portion of the nidus; and in gallstone formation in recent subjects of typhoid fever experimental evidence indicates that the Eberth bacilli, clumping in the gall-bladder, may offer nidi for precipitation of cholesterin and other products entering into the composition of biliary calculi.

A cholecystitis recognizable other than by minute pathological changes in the wall of the gall-bladder is not an essential factor in gallstone formation.

Cholecystitis is more common in women than in men, and is still more usual in childbearing women (of 115 women gallstone subjects, 99 had borne children—Naunyn). The frequency of the affection increases with age. This preponderance of frequency is evidently related to increased bile stasis, resulting from the relatively less respiratory activity in woman than in man, in the pregnant woman than in the nullipara, and in the aged than in the young.

Cholelithiasis is a very common condition. Riedel (quoted by Kehr) holds that in Germany nearly 10 per cent. of the adult population are gallstone subjects. This percentage is probably much greater than exists in Great Britain or in the United States. It is interesting and important that but a relatively very small percentage of those with gallstones ever have symptoms suggesting their presence. Kehr believes that approximately 95 per cent. remain free from symptoms; but this estimation is probably much too high, as many cases of minor transient and infrequent attacks of colic due to mild cholecystitis occur in those who never consult the physician or in whom these transient colics are wrongly diagnosed. However this may be, this infrequence of symptoms is a suggestive fact and indicates what seems now well established, that something other than mechanical irritation of the stone is required to induce the symptoms whereby the presence of calculi is recognized.

Cholelithiasis is usually latent until a reinfection of the gall-bladder determines the condition manifesting its presence.[2] If, with stone formation, the infection of the gall-bladder has not given rise to other than minor and transient pathological changes in the walls of this viscus, and adhesions are absent and the cystic duct patent, only in exceptional cases is it likely that attempted extrusion of a stone occurs. Symptoms incident to the latter commonly originate from a cholecystitis dependent upon reinfection, either newly acquired or from a pathogenic micro-organism, long latent in the gall-bladder, perhaps the original source of the calculi. There are many instances on record of typhoid cholecystitis occurring primarily a number of years after the original attack of typhoid fever.

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[1] Frerichs long ago held that gallstones originated as the result of bile stagnation, the mucus secreted by the gall-bladder favoring an acid reaction of the bile, with consequent precipitation of its salts. He was, of course, unaware of the rôle bacteria played in the formation of calculi.

[2] Cholecystitis may occur primarily without the presence of stone, as in the acute infection of the gall-bladder in typhoid fever and in pneumonia.

While mechanical irritation of the stone itself rarely originates biliary colic, it is, of course, probable that in conditions favoring stagnation of bile in the gall-bladder and prolonged distention of that viscus, such as would be induced by muscular inactivity and fasting, that transient colics occur in gallstone subjects, without a coincident cholecystitis. Usually frank attacks, or those of mild, ephemeral nature, if tending to recur, indicate infection of the gall-bladder, with its resulting inflammation. The symptoms of recognizable cholelithiasis are, therefore, usually those of cholecystitis or one or more of the latter's complications, such as pericholecystitis (adhesions), obstruction of the common bile duct, or cholangitis.

The most common form of cholelithiasis encountered is that associated with catarrhal inflammation of the gall-bladder, the result usually of a moderate grade of infection. In this there is often an abortive attempt at passage of a stone, and the stone may engage the neck of the gall-bladder or lodge in the cystic duct. The infection is transient and its degree mild. There may occur a so-called dropsy of the gall-bladder of either minor or major degree, but ephemeral almost in duration, if obstruction of the duct by a stone does not exist. In any event, with infection of mild degree and transient, the symptoms are of brief duration unless the calculus passes into the common duct, and there lodging, temporarily or permanently, by interfering with the outflow of bile, causes the usual phenomena of obstruction with jaundice. This last is the form of gallstone colic alone usually recognized by the general practitioner. With a high grade of infection, and either with or without a patent cystic duct, empyema of the gall-bladder may develop either immediately or subsequent to simple dropsy of its cavity. Empyema may occur coincident with a primary attack of hepatic colic, or after repeated seizures, with more or less long intervals of repose. If the calculus has entered the common duct, the infection continuing, there may result diffuse inflammation of the smaller bile ducts, or cholangitis.

A frequent accompaniment of repeated attacks of calculous cholecystitis, is pericholecystitis, resulting in the formation of adhesions between the gall-bladder and contiguous structures, such as the omentum, stomach, or intestines.

Apart from perforation of a gall-bladder the seat of a recent empyema with frank symptoms, a not rare effect of latent cholelithiasis with adhesions, is perforation of the gall-bladder or of one of the bile ducts harboring a calculus, with passage of the latter into a contiguous adherent organ, such as the bowel or stomach, the first recognized indication of gallstone being the voiding of the calculus by the bowel or an obstruction of the lower part of the ileum by the stone too large to pass the ileocæcal valve.

As a more remote but not uncommon effect of gallstone disease there may result dilatation of the stomach from adhesions about the pylorus or duodenum constricting its outlet; recurring gall-bladder colics even without presence of stone from similar adhesions, and gastralgia and enteralgia similarly arising. In addition to the numerous ill effects from adhesions, other not unusual remote results of gallstone disease are carcinoma of the gall-bladder, or of the common duct, or of the head of the pancreas. Hepatic cirrhosis from continued occlusion of the common duct, interstitial pancreatitis and acute hemorrhagic pancreatitis, and pancreatic fat necrosis are among the complications or more remote effects.

Catarrhal calculous cholecystitis is not only very common, but, as before remarked, is frequently improperly diagnosed. My attention was first forcibly directed to this fact long ago by some remarks of Jonathan Hutchinson.[1] In then discussing a paper by Dr. Ord concerning the more rare symptoms produced by gallstones, Mr. Hutchinson referred especially to these slight passing attacks of colic which were commonly regarded to result from stomach disorder, but in reality were due to spasm not of the stomach, but of the gall-bladder. He spoke of the importance of an inquiry for a history of such attacks in the past in arriving at a diagnosis, when later in the case more pronounced symptoms suggestive of the presence of gallstones occurred. He dwelt on the necessity of careful inquiry for symptoms which may have been regarded as too trivial to mention, and on the necessity for examination of the gall-bladder region, and emphasized the mistake so often committed by the physician in viewing jaundice as essential to the diagnosis of cholecystitis. He remarked: "Hepatic colic is a far more common event than it is generally supposed to be, and many cases escape recognition because there is a general belief that jaundice ought to be present."

This statement of Hutchinson impressed me very forcibly at the time of its publication, and caused me to be on the alert for such cases, and in the past fourteen years has been very helpful in their recognition. I have seen many that might have been viewed as simple gastralgia and as cases of recurring gastritis that were unquestionably calculous cystitis. Certain of these cases have been of a very mild type; there has been little or no rise in temperature noted, and the colic recurring irregularly at intervals of a few days is unassociated with a palpable gall-bladder. The pains, too, are often referred solely to the epigastrium, but examination may show the gall-bladder region to be acutely sensitive during the seizure, with frequently a continuance of the sensitiveness in less degree for some time after. It is especially important to be on the watch for such cases, since in certain of them in which obstruction of the duct has occurred, with resulting hydrops, empyema of the gall-bladder may develop very insidiously.

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[1] British Medical Journal, June 30, 1888.

A case of mild recurring, probably calculous, cholecystitis is now under observation in a woman, aged thirty-six years, who, apart from attacks of what she presumed to be stomach spasm, has always been in good health. The first attack occurred four years ago, and, she thought, had been induced by eating when over-fatigued. The second attack occurred seven months later, and following this they recurred about twice a year until two years ago; then their frequency increased, a seizure appearing at intervals of two or three weeks, until I saw her one year ago. The attacks occur suddenly without premonitory warnings, and bear no definite relation to the time of meals or character of food eaten. Severe pain appears in the centre of the epigastrium and to the right. The seizures have occurred through the night on three occasions only. She thought they were more usual before the menstrual period.[1] There are no special dyspeptic symptoms save, following the attacks, flatulency. The tongue is clean and the bowels regular. Vomiting is not unusual before the seizure. The attack often lasts an hour or two, and is dissipated by hot applications and the ingestion of cajuput oil in spirits of chloroform. The patient is of full habit and is very fond of sweets, and takes little exercise. Examination showed slight gastroptosis; gastric motility normal; diminished secretory activity. Examinations in the intervals disclosed slightly palpable, sensitive gall-bladder; no sensitiveness elsewhere in the abdomen. Palpation of the gall-bladder region, although carefully done, was followed by extreme soreness persisting for several days. The patient was subsequently seen once during a seizure when, although the gall-bladder was not palpable, there was marked tenderness in this region. This patient had been treated for simple gastralgia until I saw her. Stones probably at no time were voided; the attacks were of too short duration to suggest this, and jaundice has never occurred. The patient has had no seizure since the first few months of treatment. Gallstones were persistently searched for in the stools.

An interesting case recently seen of dropsy of the gall-bladder is the following: Mrs. H. H. S., aged fifty years. In September, 1901, the patient, while driving, was seized with severe epigastric pain and vomiting. The pain continued irregularly for two or three days. Some six days after the appearance of the attack I was summoned to see her. There had been no colicky pain for several days, and there remained merely a sensation of "soreness" in the epigastrium and right hypochondrium. Her stomach had been much disturbed by the remedies previously employed, and there was persistent nausea, with fetid breath and foul tongue. Although there had been a febrile reaction, the temperature now did not exceed 100° F. Examination disclosed a readily palpable and large (reaching to the umbilical line) but not hard gall-bladder, very little sensitive to pressure. It was interesting that there had been two similar though much briefer attacks occurring about six months apart some ten years before. The patient could not recall the occurrence of pain from that time until the present. Jaundice was absent in these as in the present attack. Here there was an undoubted acute hydrops of the gall-bladder, with stone probably engaging the cystic duct. The case was under constant observation for two weeks, when, but little diminution in the size of the tumor occurring, I had Dr. Neilson see her, with a view to operation. Consent of the patient was first given, but subsequently withdrawn. At the end of the third week the gall-bladder had diminished to about one-half its original mass, but was still easily palpable. She then was no longer under treatment.

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[1] It is a fact that the occurrence of pains of this sort is more common at this time, probably for the reason that, as mentioned by Kehr, the portal viscera, then more surcharged with blood, are more subject to inflammatory reaction.

In this case, as remarked, the cystic duct is probably still obstructed by a stone. The infection originating the acute seizure was evidently slight, leaving a simple dropsy of the gall-bladder. If I am right in my surmise concerning the presence of stone in the cystic duct, a cholecystotomy should have been done. This I persistently urged, resultlessly. On inquiry at this writing about her condition, the patient, who had not previously been seen for about a year, informs me that she has remained free from recurrence of colic and regards herself in perfect health; but she states it is not unusual to be conscious of a sensation of weight and discomfort in the region of the gall-bladder. An examination at this writing shows resistance and fulness here; and although, because of a recent increase in abdominal plethora, the gall-bladder cannot be palpated with distinctness, it is evidently still somewhat enlarged. I regard it probable that there are adhesions about it which later will cause trouble.

I have seen many cases similar to the two foregoing, and cite them as specimens of a class.

Two weeks ago a physician brought to me a man whom he presumed to be suffering with recurring attacks of simple gastritis. There were seizures lasting upward of a week or two, in which there were present epigastric pain and vomiting, with intervals of freedom from more than slight pain, but with present gastric irritability for several months. The patient was brought in the interval, and advice desired because of the irritable stomach. As jaundice had never been present, cholecystitis had not been suspected. An examination showed acute sensitiveness in the gall-bladder region and in no other situation. The gall-bladder could not be palpated because of the muscular rigidity and tenderness. Careful inquiry elicited the fact that the pains were as severe in the right hypochondrium as in the epigastrium. The case was clearly cholecystitis such as demanded surgical interference. The patient was immediately sent to Dr. Deaver, who acquiesced in the necessity for operation, but the patient's consent could not be obtained.

The following strikingly interesting case shows with what few symptoms calculous cholecystitis may develop and continue despite the presence of a chronically very large gall-bladder and stones in the cystic duct:

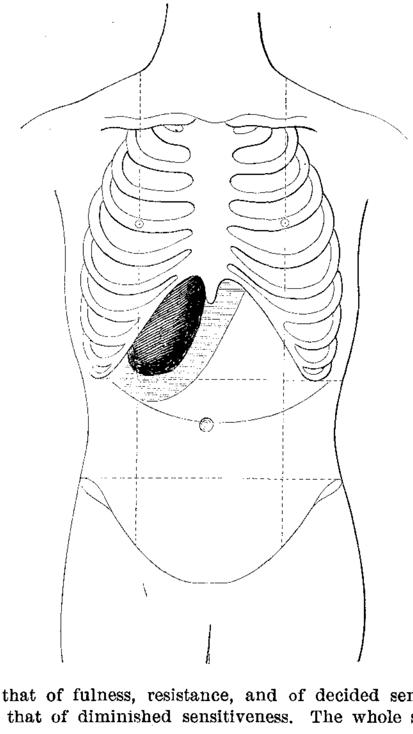
In May, 1898, I saw, with Dr. Pottberg, a woman, aged seventy years, who complained merely of a dragging sensation in the right side. Dr. Pottberg had noted the presence of a tumor, but was uncertain as to its nature, because of the absence of symptoms. The patient was otherwise in perfect health and without any indications of senility. It was only on close questioning and with difficulty she recalled that several years before she had had at short intervals a few slight attacks of what she regarded as "stomach cramp," for which she had not consulted a physician. Jaundice had never occurred. Examination disclosed a very large and apparently unadherent and but little sensitive tumor, which was plainly the gall-bladder. It caused inconvenience merely by its weight. The diagnosis was chronic obstruction of the cystic duct by stone, with the presence, presumably, of many calculi in the gall-bladder. Immediate operation was advised. This was performed by Drs. T. R. Neilson and H. C. Deaver, at the Episcopal Hospital. Section disclosed a very large and unadherent gall-bladder. It contained over one hundred stones and a great quantity of mucoid material. Two good-sized stones tightly engaged the cystic duct. These were removed and excision of the gall-bladder practised. The patient made a prompt recovery and has been in good health to the present.

The diagnosis in cases like the foregoing, examples of simple calculous cholecystitis, is readily made if one is on the alert. The history of colicky pain associated with persistent or recurring sensitiveness in the gall-bladder region, even though the gall-bladder be not palpable, should at once lead to a conjecture as to the probable nature of the ailment. A cholecystitis when occurring primarily, apart from a general infection, is very apt to be associated with calculus. Occasionally a case is encountered in which the results of a pericholecystitis, probably following several acute seizures, suggest the notion of latent calculus, although no stone may be present. In such a case adhesions resulting from attachment to the omentum, duodenum, stomach, or colon are the cause of colic or at least of soreness and discomfort in the gall-bladder region. If the gall-bladder should be patent and kinking of its duct intermittently occur, colic may thus arise in the same manner as if a stone were temporarily obstructing the duct. In such cases calculi may have been present, but been voided. I have seen several cases of this nature. In the following recently observed instance the diagnosis of adhesions was confirmed by operation. The case is a most instructive one.

In this case the presence of attacks of recurring slight colic, often associated with diarrhœa, of persistent sensitiveness in the region of the gall-bladder, of an ill-defined tumor and of interference with the gastric function, caused me to make a diagnosis of chronic cholecystitis with adhesions, probably to the duodenum. It was presumed the condition was dependent upon cholelithiasis.

L. W. T., aged forty-four years, consulted me in December, 1899. For the preceding four years his stomach had troubled him. Briefly the symptoms were: More or less discomfort at all times in the region of the stomach and right hypochondrium, especially manifest an hour or so after meals. It is more marked when in the erect posture, and is scarcely evident when lying down. No pyrosis; no flatulency. Bowels irregular, with a tendency to looseness, with pains in the gall-bladder region. These pains diffuse over the abdomen, and, following an attack of pain, diarrhœa lasting from a few hours to a day or so

FIG. 1.



Deep shaded area, that of fulness, resistance, and of decided sensitiveness to pressure. Lightly shaded area that of diminished sensitiveness. The whole shaded area, region in which crampy pain is common.

may occur. Twenty years before he had had a liver trouble. Liver was then said to be enlarged ; never jaundiced ; no lues ; no typhoid fever ; hue of skin markedly sallow ; no icterus ; puffy lower lids ; acneform eruption on face ; area of fulness and sensitiveness in the gall-bladder region and below at the situation of the hepatic flexure of the colon. Situation of tenderness especially that of the gall-bladder region. Decided sensitiveness to deep pressure if extended under the costal margin. Sensitiveness diminished laterally, as is indicated in the shading in Fig. 1. Outline of liver normal. Spleen not palpable. Inferior border of the undistended stomach at umbilicus. Knee-jerks, station, and pupils normal. Several examinations of the stomach contents : Lowered motility ; high total acidity, with subnormal HCl ; impaired starch digestion. Blood examination : Leucocytes, 7200 ; red blood corpuscles, 4,350,000 ; hæmoglobin, 75 per cent. Urine : Specific gravity, 1020 ; trace of albumin ; narrow hyaline casts ; glucose absent. Diagnosis : Chronic cholecystitis with adhesions ; chronic gastritis. Treatment : Diet regulated ; systematic out-of-door (horseback) and breathing exercises. Daily morning stomach douches with hot and cold water, sodium bicarbonate in hot solution, and weak quassia infusion and sodium chloride in the cold. Lavage, succeeded three-quarters of an hour before breakfast by sodium phosphate and sodium sulphate, of each ½ to 1 drachm ; sodium bicarbonate and salicylate, of each 10 grains. Nux vomica, nitrohydrochloric acid, and taraxacum before meals. After meals diastase, rhubarb, and ipecac. Priessnitz compress constantly worn ; later, flying blisters to the gall-bladder region.

The general condition improved markedly under this treatment, but at the end of two and one-half months, sensitiveness and fulness in the gall-bladder region continuing, I had Dr. Keen operate. The gall-bladder was fixed by adhesions to the duodenum and colon ; cystic duct patulous. The gall-bladder free from calculi ; its walls thickened. When last seen, six months after the operation, local symptoms had not reappeared and the general health was good.

The case of Dr. Pottberg narrated above is unusual in paucity of symptoms and in freedom from ill effects, the result of the long-existing gall-bladder obstruction. The original infection causing the cystitis, which led to the passage of calculi into the cystic duct, was evidently of feeble intensity and short duration, and a re-infection of the gall-bladder was prevented by complete occlusion of the duct. I shall cite another case of cholecystitis in which the diagnosis was at first somewhat doubtful, the attacks simulating those of angina pectoris. Here there existed bad aortic and mitral disease with imperfect compensation, and chronic nephritis. Although calculous cholecystitis was early suspected, and later demonstrated by the recovery of stones from the passages, an operation which was suggested to the patient six months before it was performed was postponed, because of the risk involved by the bad heart condition. Operation was performed when it was feared that a rupture of the gall-bladder might ensue. This case is related in brief outline only.

Mrs. M. D., aged forty-seven years; first seen in June, 1900. Patient confined to bed. She was then in the midst of a severe anginal seizure. There was substernal and infrasternal pain; marked cyanosis, urgent dyspnœa; very irregular heart, with similarly irregular but high pressure, slow pulse; marked aortic regurgitant murmur and presystolic and systolic (transmitted) mitral murmur. Cardiac enlargement. The liver was much enlarged; the conjunctiva slightly icteroid; albumin and casts in the urine. Apart from the severe pains above mentioned, there were attacks of crampy pain in the centre of the epigastrium, but not then in the hypochondrium. These lasted an hour or two and were associated with diarrhœa. These were not manifest when she was first under observation. The seizures were at first regarded as anginal in character, dependent upon the aortic valve disease, and this notion was borne out by the improvement and marked diminution of attacks under cardiac medicaments. Tumor of the gall-bladder was not apparent until November following, five months after she came under observation in the seizure occurring just before operation, and no marked sensitiveness in the gall-bladder region was at first evident. This had on a number of occasions been searched for, as it was suspected in the early summer that cholelithiasis was a factor in the production of the attacks. The feces were from the first sieved at intervals through the summer and autumn, but no stones were found until November. She was seen in only three attacks, two such as described above, the third in November. In this, associated with the presumed anginal pain, there was pronounced hepatic colic. The gall-bladder was then found much enlarged, and highly sensitive. Jaundice other than icteroid conjunctivæ was absent. Rigors and fever existed with the seizure. I feared an empyema of the gall-bladder and urged the necessity of immediate operation. Operation by Dr. Keen, November 18, 1900. The gall-bladder although much enlarged, contained but fifteen calculi—a few very large and a number quite small. It was distended with a mucoid material and the walls looked as if in a condition of slough. The patient, although very ill after operation, finally recovered. Despite the condition of the heart and kidneys, which render her a permanent invalid, there has been no recurrence of what were first thought to be anginal attacks.

The preceding cases, excluding that of the pericystic adhesions without stone—mentioned to show a later phase of the disease—are common examples of the calculous affection, but such as are not seldom undiagnosed by the general physician. In these cases without obstruction of the common duct jaundice is naturally wanting. Should icterus of minor grade develop—wholly unnecessary to the cholecystitis itself, however, many stones may engage the cystic duct—it is originated by the infectious process invading the common duct or the hepatic ducts and the smaller biliary channels, thus developing a catarrhal cholangitis. It is a matter of common experience in consultation, although the reason for the absence of jaundice is obvious, to be met by the query, if it is a case of gallstone, Why in all this time has not jaundice appeared? So, too, the impression often exists that the occurrence of very severe pain is an essential factor in the passage of a stone, and of continued suffering with its lodgement. That it commonly is, there is no question, and no greater acute pain in any ailment may be experienced than by the passage of a good-sized calculus through the spiral cystic duct and finally through the papilla of the common duct into the duodenum.[1] It is these cases which are the rule in which jaundice and pain are so prominent that lead to the non-recognition of the exceptions. A stone may be of fair size and be lodged for some time in the common duct, yet from its shape and from the absence of inflammation about it, allow fair passage of bile onward into the bowel, the obstruction, if any, being intermittent; then a slight discoloration of the conjunctivæ only may be present. Usually, however, jaundice of high grade follows in a few days the impaction of a stone in the common duct, which jaundice may lessen and increase as the flow of bile is alternately completely stopped and again partially established, until the stone reaches the termination of the common duct, and finally entirely and more or less permanently occludes it. It is thus usual to find considerable variation in the amount of icterus with obstruction of the common duct by calculus, unlike the case with obstruction from without as by a tumor (carcinomatous growth of the gall-bladder and of the glands of the portal fissure, or of the head of the pancreas). Here, too, another point of importance should be spoken of in the recognition of these cases of calculous obstruction of the common duct, of value in differentiating them from obstructions arising from the presence of a new-growth or pressure by carcinomatous glands. In long-continued obstructions from stone, the gall-bladder commonly, though enlarged originally, finally, by virtue of the chronic inflammatory thickening of its wall, becomes undistensible, shrinks, and is unpalpable; while in obstruction of the duct from pressure by a growth a gall-bladder tumor is usual.[2] This, which has been termed the law of Courvoisier, although it must not be depended upon invariably, is of value in diagnosis, as is shown in the following case. This case, in which the diagnosis was confirmed by operation, also illustrates one of the vagaries of cholelithiasis. It is one in which, with obstruction of the common duct by a massive, tightly adherent calculus, little preceding history of colic could be obtained. The stone was finally borne without pain, and jaundice and debility almost alone, after a few weeks of colic, indicated to the patient the presence of her illness.

Miss D. B., aged fifty-two years; referred to me by Dr. A. G. Bennett, of Mahaffey, Pa. The patient stated that she had had more or less "stomach disorder" for ten years, but her general health had been little affected thereby. The symptoms consisted chiefly of slight attacks of what she regarded as epigastric pain, lasting about a half hour and then disappearing. There would be intervals of three or four months between these spells. These slight attacks of presumed "stomach colic" thus occurred for about ten years. They were unassociated with any special gastric symptoms or with noticeable jaundice, until lately. The patient was first seen June 12, 1901. In November preceding she had epigastric soreness, with some little crampy pains in the right hypochondrium. In December there was some colic, lasting about three days, with more or less continuous slight pain, referred to the epigastrium, for two or three weeks. Pain and even soreness then disappeared, and remained absent for a week or two, following which jaundice suddenly appeared when entirely free from pain, and continued for about three weeks. With disappearance of jaundice colicky epigastric pain again recurred, but no cramp was recalled to have been present in the right hypochondrium until she was once more jaundiced, about a month before I saw her. After the first attack of jaundice the skin cleared fairly well, but the scleræ remained icteroid. Since the recent attack she has been quite persistently very deeply jaundiced, but is without pain. The only symptom of that nature is that when lying on the left side discomfort in the epigastrium is experienced. Her weight a year ago was 175 pounds; present weight, 130 pounds. The greater part of the 45 pounds had been lost since the first attack of jaundice. There are no especial dyspeptic symptoms. There are present complete anorexia, extreme mental torpor and physical debility, pruritus, and obstinate constipation. The stools are of pipe-clay hue; the urine is concentrated and of porter color. Although the skin is of a deep saffron hue, she states the jaundice tint had been more marked a week or so before.

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[1] I recall that the late George Harley taught that pain invariably accompanied chronic obstruction of the common duct by stone.

[2] But if the gall-bladder contain many calculi, or its duct is early occluded with presence of fluid from simple dropsy or empyema, this rule will not hold.

Physical Examination. Abdominal walls flaccid; with full inspiration inferior border of the right and left lobe of the liver is readily palpated. Edges sharp, firm, and even; liver somewhat enlarged; gall-bladder not palpable, certainly not enlarged, for the fingers could be depressed below the liver's edge. No sensitiveness to manipulation in the gall-bladder region, and none elsewhere save just below and to the right of the ensiform cartilage, but even here not marked. Spleen not enlarged. First heart sound enfeebled.

Blood. Several examinations (Dr. Ghriskey) average: Leucocytes, 8500; red blood corpuscles, 4,200,000; hæmoglobin, 77 per cent. Several examinations of the stomach contents: Total anacidity; the removed contents always presented the ordinary water-soaked appearance common in cases of achylia; small amount of gastric mucus. No lab reaction with milk incubated twenty-four hours. Gastric motility unimpaired.

Urine. The urine is deep golden-brown; heavily bile-stained, opaque, acid, specific gravity, 1030; well-marked albumin reaction; epithelial, granular, and hyaline casts and red blood corpuscles present.

Diagnosis. Obstructive jaundice, with stone in the common duct; past calculous cholecystitis; nephritis, the result of the chronic cholæmia; probably hepatic cirrhosis in process, due to obstruction. Patient informed of the diagnosis and operation advised. Medical treatment for removal of stone was regarded as futile to attempt.

Patient admitted to my service at the Episcopal Hospital. The attending surgeon then on duty, Dr. T. R. Neilson, saw her with me and concurred in diagnosis. Carcinoma of the gall-bladder and gall ducts was, of course, considered, but was excluded.

To diminish risk of hemorrhage on account of the high grade of jaundice, with the approval of Dr. Neilson I instituted the following preparatory treatment, which was continued for two weeks. It is such as in the case of small, unencysted calculi is sometimes of service in causing their expulsion: Daily morning stomach douching with alternate hot (sodium bicarbonate) and cold (sodium chloride) solution; one-half to one hour before each meal there was administered a combination of sodium sulphate, phosphate, and bicarbonate, in hot water, with the addition of tincture of nux vomica; diet carefully regulated; extract of pancreas as a digestant. Two daily high enemas of normal salt solution—the one hot, the other cold—were given. Under the above treatment the amount of urine, previously so scanty, increased to upward of three quarts daily. Bile appeared in the stools, but the hue of the skin was little if at all changed.

Three days prior to operation I ordered calcium chloride by enema in place of sodium chloride.

Section by Dr. Neilson, June 27th: Liver somewhat enlarged; surface firm; gall-bladder shrunken and empty. A large stone was found densely adherent in the middle portion of the common duct. It was removed with great difficulty. Its size and shape is that of a large-sized olive. It was of enormous size to have so far engaged the duct. The probability is that its accretion occurred *in situ*, where it must have been for a long period. Considering the dense adhesions about it and its bulk, it is impossible to conjecture the manner in which under the preparatory treatment instituted bile should have escaped passed it; but that such was the case was shown by the appearance of the latter in the feces, as stated.

The patient made a good recovery, and subsequently underwent treatment for removal of the conditions engendered by the cholelithiasis. Mention of this treatment is made in the latter part of the paper.

I have here brought together a small group of interesting cases recently encountered which illustrate certain phases of gallstone diseases. These cases represent simple catarrhal cholecystitis, without obstruction; with obstruction; with dropsy of the gall-bladder; with indications of beginning empyema; of pericholecystitis with adhesions the result of past cholecystitis, probably calculous; of chronic obstruction of the common duct by stone, with the usual accompanying jaundice and shrunken gall-bladder.

I shall briefly consider the recognition of such cases as these, the occurrence of which is common. Frank, outspoken cases of cholelithiasis, with severe colic, jaundice, and passage of stone, are so easily recognized that they need neither illustration nor remark. It is the frequent cases without discovered passage of stone and without jaundice that are apt to mislead. The points of greatest importance in the detection of these is to have the fact of their common occurrence in mind; to recall that however transient the duration of the self-diagnosed "stomach pain," if it is recurring the possibility of its being of gallstone origin is worth considering; to be not satisfied with the statement of the patient that the seat of pain is the epigastrium, but to have him carefully note in the attacks if it has not origin and greater intensity to the right, and if possible to examine the patient during or immediately after a seizure; then careful palpation will usually establish the diagnosis. In the recognition of cholelithiasis, among a variety of simulating conditions it is necessary to exclude simple gastralgia; gastralgia the result of adhesions, as, for instance, from past ulcer; the gastralgia of gastric and of duodenal ulcer; that of adumbilical hernia (in my experience, a not unusual source of presumed simple gastralgia). Ileus and acute pancreatitis must also be borne in mind.

Space is not here at hand for systematic discussion of differential diagnosis. I shall merely refer here to a few distinctive points which I have found of especial value.[1]

The pain of latent duodenal ulcer is apt to confuse; but it, like the pain or gastralgic seizures of gastric ulcer, commonly bears more or less definite relation to the ingestion of food, occurring immediately, or, often with definite regularity, two or three hours after eating. Other usual symptoms, among which is the locality of the pain, assist in its differentiation. Adumbilical hernia, the pain of which, as before remarked, is often mistaken for pure gastralgia and even for gastric ulcer, has its seat of intensity and sensitiveness at the site of the gripped point of omentum, the small nodule of which is commonly easily felt with careful palpation in the median line above the umbilicus. Perigastric adhesions often cause obstinate gastralgia, but they are not apt to simulate the pain of calculus except when the pylorus or duodenum is adherent to the gall-bladder, and then there is or has been usually a calculous cholecystitis. Similar gall-bladder adhesions to the duodenum, omentum, or colon causing intermittent obstruction of the bowels, with alternate diarrhœa and constipation, of which I have seen several instances, are often puzzling to diagnose. Careful interrogation as to past history of liver colic and the presence of a sensitive gall-bladder region assist materially in their recognition.

Spasm of the pylorus occurring with gastrosuccorrhœa and the pains of periodic gastrosuccorrhœa, occurring as these pains are most apt to, through the night, has misled. An interesting case of this sort I recently reported to the American Gastro-enterological Association, but it is not yet published. It is worthy of brief outline here. The patient was under treatment with several practitioners for supposed gallstones for a year and a half before being sent to me by the late Dr. Charles Wirgman.

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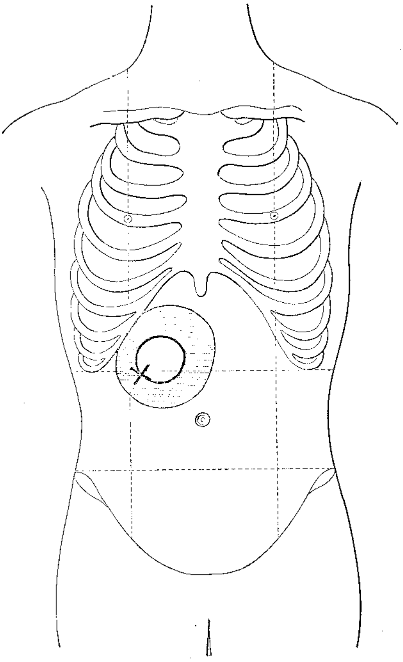
[1] I have not discussed the use of the X-ray in the diagnosis of cholelithiasis, since in the class of cases in which its assistance would be of real value—stone in the cystic duct or in the common bile duct—it cannot at present be employed to obtain results.

T. B., aged thirty years; store porter, of spare build; weight about 120 pounds; no typhoid; no syphilis. He had been the subject of attacks of sharp, cramp-like pain in the abdomen for several years. The first attack occurred three years before; then there was a lapse of one and one-half years; the seizures were very frequent in the past year and a half. Pains suddenly develop, without previous warning, in the epigastrium and to the right, in the pyloric region, shooting thence toward the epigastrium, umbilicus, and into the back; pains may last one or two days, but commonly their duration is but a few hours. Never jaundiced. Attacks occur, as a rule, between 1 and 3 A.M. Nausea is apt to accompany colic, but never vomiting unless self-induced; the vomit is then copious in amount (he states one to three quarts of acid fluid). Pain relieved by free emesis. Eats a cold lunch at noon and a hot supper, when very fatigued, at 6 P.M., retiring at 10 P.M. Twenty pounds loss in three years. He believes his previous occupation, that of a car driver, necessitating exposure and hasty, irregular eating, responsible for his ailment.

FIG. 2.



Inner circle area of fulness and firmness very perceptible when stomach inflated with air; then this area too tympanitic. The cross indicates the starting point of cramp, during the occurrence of which a prominence approximately as denoted by the shaded area appears. Pains radiate thence over the abdomen and through the right hypochondrium to the back.

Physical Examination. Mitral presystolic murmur; heart unenlarged; much dilated stomach; no tumor or sensitiveness save in pyloric (or gall-bladder) region, and then only during presence of pain. Gastric capacity, 3400 c.c. (after having been accustomed to the use of the tube); pronounced stagnation of food; sharp hyperchlorhydria. Inflation showed fulness and firmness in pyloric region—on inflation, prominence the size of a hen's egg, tympanitic on percussion and not very sensitive—a cramped pylorus. Patient seen in several seizures. Situation of pain rather suggestive of gallstone colic. Fulness and firmness in region of pylorus, a little below and to the left of normal situation of the gall-bladder. (Fig. 2.) Here there appears the above-mentioned palpable prominence, behavior of which to manipulation is unlike that of the gall-bladder. Tube introduced during seizure invariably removed a large quantity of fluid, 1 to 2 litres, which presents the appearance of gastric secretion; very little food particles evident if some hours after a meal; high percentage of HCl present; immediate lavage with sodium bicarbonate solution instantly terminates attacks. This was the case in the first seizure in which I saw him, and has been invariable since. The employment of the tube taught the patient, and thus he always was able to abort an attack.

Attacks ceased in a few months on local treatment with silver, alumnol, and bismuth, and daily lavage. The patient was under constant observation for three years, and then seen at intervals to the present. The local treatment was continued for several months and then an interlapse, to be re-begun with recurrence of seizures. At the end of about a year they ceased finally, and none have now been present for several years. At first the advisability of an operation on the pylorus or a gastro-enterostomy was considered and arranged for, but the patient would not finally consent, as his impoverished condition would not permit him to neglect his work, a family being dependent upon him for support.

This case is evidently one of past latent ulcer, followed by cicatricial contraction of the pylorus, with subsequent dilatation of the stomach. The attacks were those of gastrosuccorrhœa, with sharp pyloric spasm. The correct diagnosis was only possible by the employment of the stomach tube. As remarked, two practitioners of note prior to his consulting Dr. Wirgman had had the case under observation for one and a half years, regarding it as one of gallstone disease. No gastric examination had been made, and he had never been seen during an attack. Gallstones had been unavailingly searched for. Cases of this sort are not common, but are readily enough recognized if one is on the alert and employs modern methods of examination.

Simple gastralgia, the result of pure hyperchlorhydria, is a common enough affection, and may for a time be quite difficult to separate from mild catarrhal calculous cholecystitis. The pain, however, bears a more definite relation to food than does gallstone colic, occurring pretty definitely two to three hours after meals, but may, as in gallstone colic and in periodic gastrosuccorrhœa (which has gastralgia as an accompaniment), be most usual at night, although no hypersecretion accompanies the gastralgia. Commonly treatment instituted for the hyperchlorhydria promptly establishes the diagnosis. An illustrative case is the following:

J. M. S., aged sixty-two years; was first seen June 2, 1902. Referred by Dr. Walter Freeman. For the past one and a half years, following an attack of supposed grip, gastralgic pain, occurring at intervals of from four to five months up to last March. Since March occurring every day or so; past few weeks rarely misses a day; occurs especially when he applies himself at desk work. Six to eight years ago he had similar attacks for a short time. These disappeared entirely when he lived more in the open air. Occupation now very sedentary; takes no exercise. Appetite good when free from pain; constipation. Pain appears in the centre of the epigastrium and passes through both hypochondria to back; lasts one to three hours; diminishes in severity when he lies down; hot applications relieve. No "heartburn," occasionally water-brash; pain not especially influenced by food, but eating may relieve. No typhoid fever; no lues.

Physical Examination. Nothing noteworthy; heart normal; no sensitiveness in gall-bladder region in several examinations, or in other portions of the abdomen; stomach normally placed and undilated. Knee-jerk, station, and pupils normal.

Several examinations of the stomach contents: Hyperchlorhydria; marked atony. Urine: Trace of albumin; glucose absent; a few hyaline and granular casts; free uric acid; numerous red blood corpuscles, probably originated through irritation by the uric acid precipitated in the tubules.

Cessation of attacks was very prompt in this case under regulated diet; Carlsbad salts on rising; a full dose of an antacid three hours after meals and a combination of nux vomica, cannabis indica, and codeine before meals—codeine was taken for a short time only. At the end of a month nothing save a sodium sulphate laxative in the morning and an antacid three hours after meals were prescribed. No pain has occurred to the present (March) from two weeks after he was first seen, now nine months ago.

A similar but much more obstinate case of presumed pure gastralgia now under treatment is also of diagnostic interest. I had first suspected there might be latent cholecystitis, and examined him many times resultlessly for presence of sensitiveness in the gall-bladder region.

C. J. B., aged sixty-one years; first seen September 9, 1902. In good health up to two and one-half years ago, when epigastric pain developed. Pain occurs two to four hours after meals, and is most common at night, then there may be a much longer lapse. He fancied eating did not allay the pain, but after he had been under treatment assured me that eating is invariably remedial, pain occurring soon ceasing after a glass of milk and a few crackers. Pain in centre of the epigastrium. Seat of greatest intensity immediately below the ensiform; thence radiates, when severe, through to the back. Later, he stated, the seizures have been very severe, pain sharp and paroxysmal, but at times merely a dull ache. Duration an hour or two. No nausea or vomiting; appetite good, but fears to eat. For some months has been on starvation diet, and is debilitated in consequence. Constipation, but regularity maintained by laxatives. Best weight, 215 pounds; now, 190 pounds. Moderate drinker in the past, now for years a total abstainer; moderate tobacco user; no syphilis. Some years ago malarial fever; no other ailments; father died of liver disease; mother, aged fifty-five years, of cancer of the stomach. He feared the latter ailment, and was much depressed mentally.

Physical Examination. Knee-jerks, station, and pupils normal; stomach normally placed; no point of sensitiveness anywhere about the abdomen; gall-bladder region in many examinations totally free from sensitiveness; heart normal. Several examinations of the stomach contents: Sharp hyperchlorhydria; motility normal; three hours after a meal of steak, bread, and water, total acidity 124. HCl equal 0.365 per cent. Urine: Fair trace of albumin; hyaline casts. The patient was seen in one attack. Hypersecretion and hyperchlorhydria were found to exist. Lavage with warm sodium bicarbonate solution quickly dissipated the pain. Examination during and subsequent to the attack revealed no gall-bladder sensitiveness.

Treatment. A morning laxative of equal parts of sodium sulphate and phosphate. Before meals, cannabis indica, codeine phosphate, hyoscine hydrobromate; a full dose of antacid three hours after meals. Ordered to eat between meals when possible, then no antacid. After a week's treatment pain occurred rarely by day, but was more usual three or four hours after the evening meal, and from 1 to 2 A.M. It had lost its intensity markedly—now a dull ache, when formerly it was often an unbearably severe colic. For the first two or three days pain not markedly influenced by antacids, but subsequently and now invariably checked within a few minutes by a teaspoonful of a powder containing equal parts of sodium bicarbonate, magnesium carbonate, and oleosaccharate of peppermint. Codeine discontinued at the end of a week, and strontium bromide, cannabis indica, and hyoscine substituted for the above combination. This was again replaced in ten days by silver nitrate and hyoscyamus. Pain having entirely ceased under this and the antacid, which he now took regularly three hours after meals, all medicine save the antacid was discontinued. No pain occurred for two weeks, when a night indulgence in ice-cream, cakes, and lemonade provoked a transient recurrence. He now takes the antacid regularly, and the stomach is douched with silver nitrate solution twice a week. Dull pain now occurs rarely at night, quickly dissipated by the antacid. I regard this case as one of pure gastralgia. There are no indications of adhesions. The possibility of latent gastric ulcer is considered. Gallstone colic is positively excluded.[1]

Although acute pancreatitis, with or without disseminated fat necrosis, is an affection of comparative infrequency, its association with gallstone disease, in consequence of the latter, is well known. Acute and chronic pancreatitis may be originated through the presence of a calculus in that portion of the common bile duct near its duodenal orifice, at which point pressure may be also exerted on the contiguous pancreatic duct, interfering with the flow of pancreatic secretion into the bowel or admitting of the retrojection of bile into the pancreatic duct, as suggested by Halsted and Opie. If, as is the case in about one-third of human subjects, there is either merely the single pancreatic duct of Wirsung, or no communication between the latter the subject of occlusion, and that of the accessory pancreatic duct (of Santorini) which opens into the duodenum separably from the duct of Wirsung, conditions favorable for widespread fat necrosis and pancreatic disease exist. The pancreatitis in such cases is the result of long-continued obstruction plus infection. The obstruction offers conditions favorable for the growth of micro-organisms which have passed from the duodenum into the pancreatic duct and thus into the vulnerable organ. Without such infection it is probable that, by obstruction alone, a chronic interstitial pancreatitis may originate by virtue of the irritation resulting from the long-continued damming up of the pancreatic secretion.[1]

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[1] Since the above writing (three months) the patient has been free from attacks. The later treatment, which has been most beneficial, is courses of alumnol and silver used through the stomach tube.

Without cholelithiasis, and without such obstruction of the pancreatic duct, it is likely that infection of the pancreas from the duodenum, causing acute or chronic inflammation, may occasionally occur. Such a condition may be especially looked for in cases of chronic gastritis of bacterial origin. In several cases of chronic gastritis which I have had under observation in which inflammatory changes in the secretory glands exist, presumably the result of infection, there were indications of chronic interstitial pancreatitis which I regarded as due to a similar transmitted infectious process.

The following most instructive case is in all probability one in which acute pancreatitis was so produced. It is of extreme interest, since the diagnosis by the attendants, who had been misled by the previous history, was at first that of collapse from perforation the result of a gallstone, and finally that of impaction by a biliary calculus without perforation. This case forcibly illustrates the difficulty sometimes encountered in differentiating between acute pancreatitis and gallstone disease. Briefly the points are: There had been two attacks of acute gastritis, in which I saw him; following which gastralgia appeared. There was a chronic subacid gastritis (with erosions), probably from mouth infection. About two years after the second attack of acute gastritis, while in another city, there suddenly developed overwhelmingly severe pain in the abdomen, followed by collapse. The diagnosis of his numerous attendants was as stated above. Section demonstrated entire absence of cholelithiasis or any of its consequences, and the presence of some old adhesions and of recent lesions such as are indicative of acute pancreatitis. The case is as follows:

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[1] See a paper by Opie, The Relation of Cholelithiasis to Disease of the Pancreas and to Fat Necrosis, THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, January, 1901.

C. M. L., aged forty-seven years; plethoric habit; weight, 210 pounds; sedentary occupation; very little exercise or fresh air; indiscreet in diet; moderate alcoholic indulgence; tobacco in excess; no lues. Attack of acute gastritis in the early part of 1897, in which I attended him in consultation with the late Dr. J. M. Da Costa. A similar attack a year later, in which I also saw him. On each occasion the symptoms were those of a general infection, with a local expression in the stomach. Vomiting had occurred at the early part of the attack; stomach-contents foul smelling; free HCl and the ferments absent; excess of organic acid; fever; epigastric sensitiveness and pain; congestion of the base of the right lung in each seizure. Urine contained albumin, hyaline and a few granular and epithelial casts. Following the first seizure of this sort there were recurring spells of mild gastralgia, in duration an hour or two and occasionally longer. Pain rarely severe, and commonly easily checked by lavage and the use of codeine. Repeated physical examinations gave no indication of the pain being other than gastralgic. Several examinations of the stomach-contents in the interval during two years always showed: Invariably absence of free HCl; pepsin and lab ferments absent; the pre-enzymes in traces only; acidity low (10 to 20). Blood-stained bits of the mucosa occasionally found in the wash water, presenting the usual appearance of erosions. Gastric motility normal save during the acute seizures. Stomach undilated and normally placed. The urine in the interval of attacks of acute gastritis was practically normal. There existed a severe type of pyorrhœa alveolaris. This had been present for years, and had never received adequate attention. Bacterial cultures from the gingival margins showed among many other organisms the staphylococcus pyogenes albus and aureus.

Diagnosis. Attacks of acute gastritis intercurrent with chronic glandular gastritis; gastric erosions; gastralgia; the gastritis probably the result of the alveolar pyorrhœa. The gastralgia was regarded as probably of nervous origin, although the possibility of its arising from perigastric adhesions the result of long-continued gastritis was noted. Gastralgic pain tended to recur despite medical treatment, and after the second attack of acute gastritis the patient was persuaded to stop work and take a six weeks' summer holiday. Gastralgic pain had then been annoying him considerably. This immediately and entirely ceased from the day he abandoned business. He went into camp in Canada; tramped, fished, and shot, eating ordinary camp food and frequently getting wet. This exposure, with rough food and a great deal of active exercise, in the nature of things should have been productive of attacks of gastralgia had they been due to adhesions. Their non-recurrence and their complete absence for some months after his return, despite active tennis, suggested these pains were of nervous origin. The following winter (1898–99), despite close application to business and general unhygienic living, he was in better health than he had been for a long time. The succeeding summer his outing was brief and his application to work close. In the autumn following (1899) I saw him in two attacks of presumed gastralgia; in one he was confined to bed for twelve hours, and the pain was quickly relieved by lavage. There had been diffuse gastric colic, associated with stagnation of food and decomposition of the ingesta. The cardia was in spasm, so that the stomach tube was entered and withdrawn with difficulty. The second attack immediately followed a dinner party. The pain had been of a half-hour's duration when I saw him in my office, and was instantly checked by lavage.

Some months later, while in another city, he had an attack altogether unlike any of the preceding. There suddenly appeared severe abdominal pain and vomiting. On the arrival of his physician he was in a state of collapse. There was stated by his attendant to be "cyanosis, shallow thoracic breathing, diffuse abdominal pain and tenderness" (the pain and tenderness thought by the attendant to be largely epigastric and hepatic), "a rapid, thready pulse and cold sweat." Calculous cholecystitis, with rupture of the gall-bladder, perforating duodenal or gastric ulcer, it was reported were thought of, but the condition was so alarming that little examination was immediately possible to determine the exact diagnosis. It was with difficulty that he was rescued from the collapse which had so suddenly occurred. For three days following it was reported that he was so ill that he was unconscious of his surroundings. There were fever (101° to 102½° F.) and incessant hiccough for about a week. There were "congestion of the base of one of the lungs" and "albuminuria and a slight biliuria." In addition to his regular attendants he had been seen by several medical consultants of eminence and a surgeon of equal note, and it was unanimously agreed that the case was undoubtedly gallstone, with possible perforation, and if not this, at least impaction of a large stone in the duct. Operation was regarded as essential, but his condition did not warrant it being undertaken until about three weeks after the onset of the seizure. The diagnosis of either perforation by a gallstone or of impacted calculus was arrived at by the attendants through a process of exclusion, and because the previous history had made it certain to their minds that the various gastralgic attacks and those of acute gastritis which had occurred in the past were undoubtedly only expressions of the vagaries of cholelithiasis. Upon this there was apparently no dissension, although in correspondence with the attending physician I urged that there had been nothing in the nature of the previous seizures to suggest this; that gallstone colic had then been carefully excluded.

Section disclosed the following: Patient's abdomen very thick walled; adipose tissue at least three and one-half inches; gall-bladder and bile ducts normal; no present or past indications of gallstones. Instead there existed evidences of recent peritonitis; a mass of adhesions binding the hepatic colon, stomach, and pancreas, some recent and some old. Omentum thickened. Pancreas adherent to stomach; *inflammatory mass about and involving an enlarged pancreas.* The operation had to be conducted with great celerity because of the patient's condition, and, it was stated, little time was permitted for examination after separation of adhesions. It was, however, noted that the pancreas was much enlarged, and this enlargement at the time was thought to be the result of a malignant growth. The subsequent history of the case has disproved this. The patient is now living and in good health. As I remarked, I have no doubt that the attack was one of acute hemorrhagic pancreatitis—an acute pancreatitis of infectious origin perhaps engrafted upon a chronic inflammatory condition of this organ. The infection originating the pancreatitis probably bore some relation to the long-existing chronic glandular gastritis, which itself was presumably due to the continued passing of pus organisms from the mouth into the vulnerable stomach.

The origin of the old adhesions about the stomach is in doubt. The two attacks of gastritis spoken of were unaccompanied by indications of perigastritis—at least the local inflammatory conditions were not then regarded as sufficiently severe to suggest this. Subsequent to operation the patient recalled that years before he had had a fall, striking the abdomen, and it was thought possible that adhesions might have so arisen. That these old adhesions about the stomach bore some relation to the earlier attacks of gastralgia I have no doubt, but that they were the sole cause of this I question, for the reasons cited.

This case well illustrates the difficulty that may attend the separation of an attack of acute pancreatitis from presumed perforation by a gallstone, and indicates how a previous history of epigastric pain may mislead even the most skilled diagnostician. But for such a history, which at once led to the diagnosis of gallstone disease by his attendants, in consequence of which the question of pancreatitis was not, I believe, even considered, it might have received attention. The sudden onset, when in presumed good health; severe abdominal pain; vomiting of at first the contents of the stomach and, later, copiously of bile; the rapid appearance of collapse, continuing, with the occurrence of obstinate hiccough and the like, as detailed, were very suggestive.

THE TREATMENT OF CHOLELITHIASIS. There is no solvent for gallstones in the gall-bladder or in the bile passages. The employment of sodium cholate, of sodium succinate, and sodium oleate; of the more rational sodium salts presumed to have some cholagogue effect, such as the phosphate and sulphate; of the old French mixture of ether and turpentine, and of chloroform, is no longer favored by the intelligent physician with the expectation of dissolving calculi in gall-bladder or bile ducts. I confess that some fifteen years or more ago I was an adherent to the contrary notion, and, as a disciple of George Harley, for several years religiously saturated my gallstone cases with sodium cholate and the like, with vain expectation of promoting solution and expulsion of calculi. After a few years of increasing familiarity with the pathology of cholelithiasis, aided by the teaching of the operating and post-mortem table, I became convinced of the futility of the solvent treatment. Treatment of active cholelithiasis is now justly very largely abandoned to the surgeon. There is no medical treatment for persisting dropsy of the gall-bladder, or for obstruction of the cystic duct; for seropurulent or purulent inflammation of the gall-bladder, or for stone long lodged in the common bile duct. But it is unnecessary, and the surgeon is usually unwilling to accept that we shift to him the cases of simple calculous cholecystitis, presumably without obstruction—cases such as those first cited—until after a fair effort to dissipate the ailment without the knife. If the patient is in a position to have the best done for him at home, and, if necessary, to visit Carlsbad for a season or more, even though the attacks at first show a tendency to fairly persistently return, should they remain mild in character and should enlargement of the gall-bladder not be evident on repeated examination, nor persistent sensitiveness be present, it is proper that such cases be not hurried to the surgeon. Medical treatment, however, is not to be instituted with the view to the solution or extrusion of calculi, but with the object of both controlling the inflammatory catarrhal process in the gall-bladder and of preventing its recurrence, trusting with its abatement that the calculi present will no longer create trouble. It seems probable, as before remarked, that gallstones do not mechanically develop cholecystitis, whereby their presence is manifest. Our present knowledge of the pathology of that affection is against this old notion. It is also likely that only very rarely does attempted expulsion of a stone from the gall-bladder occur without a coincident cholecystitis, the latter, as Kehr remarks, being actually the force that initiates the movement of the stone. Cholecystitis is a very frequent condition, and is far more frequently latent than active, for with presumably almost 10 per cent. of adults carrying these concretions, but possibly somewhat more than 5 per cent. of this very large number have recognizable indications of their presence.

Briefly, I should place the necessity for surgical interference in these cases of catarrhal cholangitis, without presumed obstruction of the duct, in this wise: If the patient is well-to-do, and can spend a season or two at Carlsbad; if there is no strong family history of cancer;[1] if the cholecystitis is recent and of mild type and shows a tendency to no persistent recurrence after some months of treatment, surgical intervention, although not improper, may be regarded as yet unnecessary. Should there be an old history of recurring pains, and the attack for which one is consulted suggests cholecystitis, I regard the case as immediately surgical, for in such a one further recurrences are probable, and the likelihood of pericystic adhesions is strong. Concerning the question of necessity for surgical intervention in cases of stone in the common duct, my view is that these should be operated on no later than a month after a jaundice has developed; since with prolonged obstruction, and with so few if any adequate means at hand to promote passage of the stone, and with danger of complication so vital, it is unmedical and unscientific to delay. A stone sufficiently large to completely obstruct the duct, after a month's impaction, in the vast number of cases, is lodged permanently unless removed. No amount of oil or Carlsbad treatment here after this lapse is likely to be efficient. Although jaundice may be temporarily diminished, the stay of the stone will be uninfluenced, while, in consequence of the persistent back-pressure, pathological alterations in the liver structure are soon in progress. Hepatic cirrhosis is a not uncommon result of long-continued biliary obstruction, as are, under the influence of the cholæmia, changes in the kidney. In the case of Miss D. B. related, although the stone had probably not been carried many months, the liver already displayed indications of cirrhosis, and a pronounced nephritis was present, of presumed cholæmic source. This case well illustrated the utter futility of medical treatment in a subject with stone of large size fairly long lodged—the inutility of any drug, however active, as a stone solvent or as an aid to stone expulsion. In this case accretion of the stone probably had occurred in the common bile duct, since it may be regarded as too massive to have passed the cystic duct without more marked symptoms than were originally present.

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[1] The development of carcinoma of the gall-bladder is favored by the presence of gallstones; about 14 per cent. of gallstone subjects develop cancer of the gall-bladder (Kehr). Courvoisier found gallstones present in 87 per cent. of cases of carcinoma of the gall-bladder, and Ames in 95.4 per cent. (quoted by Kehr).

Other and not remote dangers of prolonged obstruction of the duct to be mentioned are perforation of the duct and pancreatic disease—pancreatic fat necrosis, and acute or chronic pancreatitis. Pancreatitis may follow very shortly after the lodgement of a stone in a situation to obstruct the duct of Wirsung. Cases are reported in which, as stated by Opie,[1] decided pancreatic lesions have followed forty-eight hours after the onset of symptoms of impaction of the calculus, a period too short even for the production of marked jaundice. Although such cases are unusual, the later occurrence of pancreatitis in consequence of obstruction is less so, and is, with other risks, one which physicians must consider, not too long delaying surgical co-operation.

The one object in the treatment of simple cholecystitis is to endeavor to render quiescent the inflammatory process in the gall-bladder and prevent, rather than favor, the passage of a calculus from that viscus. During and following the acute seizure active purgation must be avoided, as must the use of all measures which are presumed to have cholagogue effect. Under no circumstances, even in the quiescent period, must massage of the gall-bladder or of the gall-bladder region be attempted with the idea of diminishing the size of this organ or of assisting to expel its contents. Should a gall-bladder be palpable after an attack, its duct is in all probability obstructed, and the only treatment is surgical. The risk of rupture of an inflamed gall-bladder through massage is not slight. Morphine must be employed with caution in acute cholecystitis because of the danger of masking the local inflammatory condition, and because of the effect the drug may have upon the stomach, acting, as it so frequently does, to later enhance the already existing gastric irritability. Nausea and vomiting and pain, save when a stone is presumably already in passage, should be controlled by other means. *Persistent* nausea and vomiting in simple acute cholecystitis probably indicate that the calculus has already entered the cystic or common bile duct, and even if such is not the case, vomiting reflexly favors the engagement of a calculus.

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[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, January, 1901.

In the treatment of a case of cholecystitis in the interval it is important to ascertain the condition of the motor and secretory gastric functions, and to appropriately treat any existing abnormality. Chronic gastritis frequently accompanies cholelithiasis, and when the former is of mycotic origin it favors the occurrence of the latter. The hygiene of the mouth should likewise receive attention, that infection of the stomach from diseased teeth or gums be obviated. It is held that gallstone suspects, irrespective of the condition of the motor or secretory gastric functions, should take food at short intervals, with the idea that stasis of bile in the gall-bladder and resulting colics be thus rendered less likely. Clemm, of Darmstadt,[1] for instance, while stating that all catarrhal gastro-intestinal affections should be thoroughly treated, suggests that "there should not be more than two or two-and-one-half-hour intervals between meals, with milk between at least twice," and that "a glass or two of milk should be taken before rising in case of the slightest suspicion of stoppage of the flow of bile," and that "the earliest and last meal should be principally albumin and fats;" and Kehr, who is also an advocate of frequent feeding, urges that a meal be taken on retiring. While it is true that during fasting the gall-bladder acts as a reservoir for bile, and that frequent feeding favors its direct flow into the bowel, the risk of increase in gastric atony, should this exist, and of aggravation of the gastro-intestinal catarrh so often present, with transmission of this catarrhal process from the duodenum to the bile duct, is sufficiently great to necessitate caution in the matter of very frequent meals. Feeding in this way is only indicated in those with well-preserved gastric motility or with hypermotility, and especially in such cases with hyperchlorhydria. The interval of meals should thus depend altogether upon the ability of the stomach to empty itself and largely upon the condition of the secretory function. In cholecystitis, as there is no interference with the passage of bile and pancreatic juice into the intestine, and as the gastric secretory power may be normal, no very special diet is essential.

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[1] Wiener med. Presse. Quoted in the Journal of the American Medical Association, September 6, 1902.

The food must, of course, be plain and easy of digestion; high seasoning must be avoided and rich entrées and pastry tabooed. No digestant is indicated if the gastric secretory power is normal and proper insalivation of food is enjoined. In obstruction of the common duct, in which there may be coincident obstruction of the pancreatic duct, as with the stone in the diverticulum of Vater, the diet should be largely albuminous, and food into which the composition of starch enters little if at all allowed. If substances containing starch are eaten, the starch should be either first dextrinized or diastase should be administered. Pancreatic extract can be of little real value as a digestant if the gastric secretory function is normal or heightened, as destruction of its ferments in the stomach is certain; but with subacidity or anacidity, fairly frequently present in these cases, pancreatic extract may be freely given with meals to serve the triple purpose of an albumin, starch, and fat digestant. It is then probably of more value, by virtue of its contained steapsin, than is the conjoint use of papain and diastase alone, which later are practically without action on fats.

Gastric atony accompanying cholelithiasis is best treated by daily morning stomach douching with alternate hot and cold water; weak sodium bicarbonate or Carlsbad salt solution in the hot water and sodium chloride in the cold—the last is preferably a weak quassia or calumba infusion. In all cases there is prescribed sodium sulphate either alone or in one of the Carlsbad combinations, such as the Carlsbad salt in powder or in crystal form,[1] or Carlsbad water, or, what I prefer and have used for years, a combination of sodium sulphate and phosphate or of sodium sulphate, phosphate, and bicarbonate. The salt is administered in rather hot solution on rising, subsequent to the lavage if this is practised; a small quantity of bitter infusion, such as quassia or calumba or of the tincture of nux vomica, taken coincidently, renders the saline draught better borne by the stomach and stimulates its passage into the bowel. Movement and breathing exercises should be taken after its ingestion, and no food is permitted for upward of an hour afterward. The solution should be drunk slowly and either in sips or in small draughts, at a few moments' interval, as its passage from the stomach is thus more rapid than if it has been hastily gulped. If the bowels are sluggish, and a tendency to looseness is not set up by the Glauber salt, I give a similar combination in smaller dose a half-hour before lunch and dinner, and on going to bed if food is not then taken. When sodium sulphate or phosphate is used more often than once daily and long continued, the dose should not ordinarily exceed gr. xx to ʒss of each. If hyperchlorhydria exists, sodium bicarbonate is omitted from the early morning combination, and a full dose (gr. xl to gr. lx) given either alone or in combination with a small quantity of Glauber salt or sodium phosphate three to four hours after the preceding meal and not later than one-half hour before the following one. The fluid extract of taraxacum or the succus taraxacum in 1-drachm to 2-drachm doses is coincidently administered.

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[1] The crystalline salt is more laxative, containing a much larger percentage of sodium sulphate than the salt in powder form.

Whatever the condition of the motor or secretory function of the stomach, notably good results are often obtained in cases of cholelithiasis by the daily morning use of strong sodium bicarbonate solution through the stomach tube, in the following manner: A funnelful (about 750 c.c.) of water at 105° to 110° F., containing two heaping teaspoonfuls of sodium bicarbonate, is passed into the fasting stomach, the intragastric extremity of the tube engaging the cardia (approximately sixteen inches); the funnel is held some distance above the head to obtain a douche effect. The stomach region from the fundus to the pylorus is massaged as the fluid enters, and subsequently for two to three minutes, after which the solution is siphoned off, and the stomach is douched once or twice with cool normal salt solution. The effect of soda thus used is beneficial in promoting a free outflow of bile into the bowel and in diminishing the viscidity of the bile. Should the bowels be acted upon too energetically, the stay of the soda solution in the stomach is lessened or the quantity of soda used diminished, and no saline laxative follows its employment.

Abundant out-of-door exercise, tennis, horseback-riding, and the like, except in case of very recent active symptoms, is insisted upon. Too energetic exercise is not permitted when there is persistent sensitiveness in the gall-bladder region or when there is a history of frequent recent attacks of colic. Energetic breathing exercises, general light massage, walking, hill-climbing, are all of utility in latent cases in stimulating the hepatic function and in obviating bile stasis.

If the trouble recurs despite carefully carried out treatment on these lines, and the patient is well-to-do and disinclined for operation—necessity for which not seeming too imminent—I advise a sojourn at Carlsbad. There, where business and family cares and social duties can be laid entirely aside and the life of the place becomes a pleasant incentive to recover health, much more may be accomplished on a similar regimen than at home. A woman patient with chronic cholecystitis whom I sent to Carlsbad three years ago, and who was rid of recurring attacks by two seasons there, received approximately the following course of treatment: Before rising in the morning it was customary to sip a tumbler of hot Sprudel water. Immediately after rising gentle exercise was taken, such as walking about and dressing, and in fifteen minutes a second tumbler was slowly drunk, followed by a third a half hour later. If the patient's strength permitted there was a considerable interval (upward of an hour) before breakfast, during which a rather active walk was indulged in. Breakfast was commonly very light, such as toasted bread and tea, the latter without milk and sugar. This was followed in two hours by a more substantial repast—minced chicken on toast. Two hours later minced ham or chicken, with a purée of pea, potato, or spinach, was eaten. Three hours subsequently two tumblersful of cold Sprudel water were taken, with a lapse of a half-hour between, and in three-quarters of an hour after the second a meal similar to the last mentioned was indulged in. A tumblerful of Sprudel water was finally drunk before retiring. There were in addition the inevitable peat poultices applied to the hypochondrium. A good deal of out-of-door exercise was insisted upon. A number of small gallstones were voided while at Carlsbad. Quite a similar line of treatment, substituting the Priessnitz compress for the peat poultices, had been previously attempted here, but without much result, largely owing to the lack of earnest and persistent co-operation of the patient.

The object of treatment in these cases of calculous cholecystitis is not to promote expulsion of stones, but to dissipate the underlying catarrhal process and prevent its return, and, by maintaining persistent fluidity of the bile, to obviate further stone formation. Under the Carlsbad treatment and its modifications, however, small stones often are voided, leading the patient to imagine that he is thus rid of his trouble. If these calculi have not already engaged the cystic or common duct their passage from the gall-bladder is rather an unfortunate than a favorable circumstance, since the extrusion into the duct of a calculus too large to be readily if at all passed may be similarly favored.

The treatment of recent cases of stone in the common duct belongs to the physician, but for a short time only. Frequently the stone is passed into the bowel, entirely without treatment, a few days after the onset of colic. In other cases the calculus may linger in the duct despite all that is done to effect its dislodgement. In these cases of obstruction of the common duct, after the acute symptoms of obstruction have passed, I am in the habit of employing the sodium laxatives above mentioned and of resorting to the intragastric douche, with warm sodium bicarbonate solution and of high enemas of hot and cold normal salt solution, as before detailed, both for the purpose of reflexly favoring the onward passage of the stone into the bowel and of allaying the accompanying catarrh of the bile passages.

Formerly I was much inclined to the employment of olive oil in obstructions of the common bile duct by stone, and for several years used it rather freely, with results which apparently justified my confidence in this remedy. In recent years I have employed it but little, largely because of the constantly met with repugnance of patients to the remedy and because of the difficulty in its retention by the stomach when administered in the full doses necessary—and also, perhaps, because I became skeptical of its utility, cases not infrequently occurring in which passage of the stone, because of its size, could not be thus influenced. In recently reviewing my notes of several cases of common duct obstruction in which I employed oil some years ago, and in which there followed cessation of colic and passage of calculi of fair calibre (in one case a triangular-shaped calculus the size of a beechnut was voided), I questioned if it was wise to entirely abandon this remedy. Naturally oil can be of no utility in case of massive calculus, but, considering the paucity of our remedies, it is worthy of at least brief trial in recent cases of obstruction of the common bile duct, for if it can be of no utility, its employment is unattended with harm. In searching for an explanation for the mode of action of the oil, presuming the result sometimes obtained is not entirely a *post hoc* one, it occurred to me some years ago its utility might rest on a sound therapeutic basis,[1] and that there seemed reason to suppose the effect attributed to the oil might be due rather to a product of its decomposition, glycerin. With unobstructed pancreatic flow it is not improbable that by virtue of the fat-splitting ferment steapsin a portion of the oil is disassociated in the duodenum into its components, fatty acids and glyceryl, and that nascent glycerin thus formed exerts in the duodenum an effect somewhat analogous to that which takes place when glycerin is introduced into the rectum—withdrawing water and causing hyperæmia, and causing irritation of the afferent nerves of the part with which it comes into contact, and thus leading to active reflex peristalsis. I believed that a similar effect might result from the presence of nascent glycerin in the duodenum as occurs from the introduction of glycerin into the rectum for its cathartic action. In my paper on this subject I remarked, in effect, that in addition to the active contraction which might be caused by the presence of glycerin in the duodenum, and which of itself would probably tend to contraction of the bile ducts and perhaps of the gall-bladder, the power of diffusion possessed by glycerin would perhaps enable it to enter the ductus communis, and even the hepatic ducts and the cystic duct, producing a similar depletion of the vessels there, and reflexly exciting the muscular fibres of these ducts to contraction. In consequence of this action an outflow of diluted bile would be favored which I thought would assist in expelling the stone. I said that since no other explanation is at hand as to the efficiency of the oil in these cases, and that, as is well known, glycerin is normally so formed from it in the bowel by aid of the fat-splitting ferment of the pancreas, and since it is probable that glycerin can produce the effects attributed to the oil, it may be not unlikely that these are due to glycerin thus formed. Rosenberg about this time reported he had found by experiments on dogs with gall-bladder fistula that large doses of olive oil greatly increased the quantity and diminished the consistency of the bile excreted.

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[1] See my paper, A Suggestion as to the Action of Olive or Cottonseed Oil in Gallstone Colic: Observations on the Use of the Oils, and Report of Cases. The Medical News, November 23, 1889.

After the publication of my paper suggesting this theory of the action of the oil, glycerin was suggested in the treatment of cholelithiasis. If the utility of glycerin is in the manner I proposed, unless it could be immediately brought into contact with the duodenum without a stay in the stomach it would be useless as a remedy. Some trials which I later made with it in impacted gallstone were resultless.

Olive oil employed in the manner outlined in my paper is certainly worthy of a trial in recent cases of common bile-duct obstruction by stone.

Preparatory Treatment before Operation in Cases of Obstruction of the Common Duct by Stone. Although this subject belongs rather to the surgeon, a few remarks concerning it here are apt, because of its extreme importance. It is well known that with cholæmia resulting from persistent obstruction of the common duct, and especially in cases in which there is coincident disease of the pancreas, there often exists a malignant hemorrhagic tendency which, unless strenuous efforts are made to overcome it, may readily terminate life after operation.[1] In order to obviate as far as possible this condition an effort should be made to re-establish a flow of bile and pancreatic juice into the bowel and to assist in the elimination of the bile already in the blood and tissues. The line of treatment suggested in cases of impaction is of value here. I do not refer to the use of oil, but to the employment of daily lavage with hot soda solution and to the use of high enemas of hot and cold water, as detailed in the case of Miss D. B. These last in former years were in vogue in the treatment of simple catarrhal jaundice, and are often of singular efficiency in promoting reflexly a flow of bile into the bowel. Lavage with soda solution acts similarly and perhaps also directly by diminishing the duodenal and bile-duct catarrh. The laxative sodium salts are also employed for a similar purpose, as outlined previously in discussing the treatment of cholecystitis in the interval. With these, taraxacum in full doses is prescribed. A morning laxative dose only of sodium phosphate and sulphate may be administered, and nitromuriatic acid and taraxacum given a half-hour before the noon and evening meal and at bedtime. The results obtained in the case of impaction previously cited (Miss D. B.) under this treatment were quite remarkable, considering how firmly the large calculus was fixed in the common duct by adhesions. Bile appeared in the stools, and the amount of urine was enormously increased. If with this treatment the stools remain persistently clay-colored, there may be reason to suppose that the obstruction is other than by a calculus. To also assist in the elimination of bile by the urine, when the remedies suggested have had little effect in directly promoting its flow into the bowel, it is worth while to employ benzoic acid or one of its salts—sodium or ammonium benzoinate. However its mode of action, that benzoic acid has an effect in hastening the resorption of bile from the tissues, and its excretion is well established.

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[1] This hemorrhagic condition may be related to pancreatic hemorrhage occurring in many cases of acute pancreatitis. There is good evidence to believe that although the cholæmia is an important factor, it is not the sole one in the development of the tendency to bleed after operation, so often encountered in gallstone cases. Mayo Robson states that his experience in increased number of operations on deeply jaundiced subjects, teaches him that there is much less danger of serious bleeding in patients jaundiced from gallstones than in those in which the jaundice depends upon pancreatic disease.

For a few days prior to operation, after discontinuing the above medicaments, calcium chloride should be administered, with a view to heightening the coagulating property of the blood, and if necessary, during or immediately subsequent to operation, parenchymatous injections of gelatin. The sterile solution of gelatin should always be at hand for immediate use.

It is highly important that, following operation for chronic obstruction of the common bile duct, treatment be instituted to obviate, as far as possible, the ill effects of the long-standing bile stasis. The imminence of hepatic cirrhosis in these cases must be borne in mind. Apart from this grave condition there usually exists thickening of bile ducts and often a chronic gastroduodenitis. In the liver structural changes have likely occurred, due to the long-existing back-pressure of bile and, often, to coincident infection. Even if not structurally damaged, liver cells long accustomed to the passage of bile into the lymphatics do not readily assume normal function, and despite the removal of obstruction and free passage made for the bile, bile still, for a more or less lengthy period, finds its way into the circulation. A continued observation of these cases is, therefore, of prime importance, in order to correct the gastric and bile-duct catarrh, to obviate further bile stasis, and thus to restore the liver, as far as possible, to normal function. The state of the kidneys must also be carefully looked to.

The after-treatment should be largely on the lines already laid down for the management of chronic cholecystitis. In addition, mention must be made of the utility of certain other remedies in cases in which the jaundiced hue of skin persists. Here, apart from the measures cited for the treatment of the catarrhal condition, and in addition to attention to general hygiene, exercises, baths, and local applications, I have found the persistent use of ammonium chloride and taraxacum of great service. I often alternate courses of ammonium chloride with nitromuriatic acid. My usual mode of administering these remedies is to employ a morning laxative of sodium sulphate or phosphate and to give diluted nitromuriatic acid (10 to 15 minims in taraxacum, fluid extract or taraxacum juice, 1 to 2 drachms) before the midday and evening meal and at bedtime. A course of ammonium chloride (grs. xv–xx, three times daily) is alternated with the acid, and this salt is similarly exhibited in taraxacum, but is administered from one to three hours after meals.

PERITONITIS OF THE DUODENAL REGION.

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We may consider the various peritonitides as having regional significance. Thus, peritonitis of the pelvic region, or of the right iliac region, present peculiar features by which they have long been recognized. It is with chronic peritonitis of the duodenal region, or, more properly speaking, with the plastic adhesions following peritonitis of this region, that this paper will deal, with the view of calling attention to certain peculiarities of this form of abdominal disease.

By the duodenal region we shall understand that portion of the abdomen, bounded posteriorly by the descending limb of the duodenum, the inner aspect of the upper pole of the right kidney, and the common bile duct; to the inner side, by the mesocolon; to the outer side, by the gall-bladder and liver; above, by the liver and gastroduodenal junction; and in front, by the hepatic flexure, border of the liver, great omentum, and abdominal wall. This space, which we may designate as the duodeno-hepatic fossa, is, next to the pelvis and right iliac fossa, most prone to peritonitis. Every surgeon is aware of the frequency with which he encounters the results of peritonitis in this region, in the adhesions which he so often discovers in attempting to expose the duodenum or the common bile duct.

Although there are peculiar features which characterize acute peritonitis of this region, it is rather the result of such an acute inflammation, which gives rise to the chronic disturbances which are less well understood, and which are of sufficient frequency and importance to merit the attention of the surgeon and internist.

The causes of plastic peritoneal deposits in this region are about the same as the causes of acute peritonitis. The three conditions which deserve pre-eminent mention as causative factors are cholangitis; cholecystitis, with or without gallstones; and duodenal ulcer, with or without perforation. For the completion of the etiological category,